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RESEARCH ARTICLE

CAUSES OF INCREASED MEAN PLATELET VOLUME IN MANGALORE REGION

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Key words:-
MPV, Laboratory, WBC,Glucose,Biochemistry.

Abstract

Objective:To find out common causes of high Mean Platelet Volume (MPV) in a pronounced clinical laboratory in mangalore region.

Method: The present study has been conducted in a pronounced laboratory in India.

A retrospective analysis of the results obtained from the chemical laboratory for cases of variance in MPV value has been carried out on 178 patients attending both IPD and OPD.

Result: The study showed that out of 1000 samples,178 patients showed high MPV(>=9).Among these 178 patients males were more as compared to females.28% of the patients had high total WBC count,35.3% had elevated glucose , 12.9% had abnormal lipid profile and 10.1% showed high CKMB values. However 37.6% patients showed no abnormal biochemistry.10 patients showed low MPV(<6.5) which had no significance.

Conclusion: A positive correlation has been found in our study between high MPV and abnormalities in haematological and biochemical values.

Introduction:-

Platelet disorders are relatively common in the general practice of medicine. With availability of automated blood cell analyzers new indices related to platelet count are also being estimated. Most important parameters among them are platelet crit (PCT), mean platelet volume (MPV) and platelet distribution width(PDW)[1]. Platelet activation leads to changes in platelet shape with increase in platelet swelling leading to an increase in MPV and PDW.[2] Mean platelet volume (MPV), the most commonly used measure of platelet size, is a potential marker of platelet reactivity. The automated cell counter, however, provides an MPV on each whole blood sample that is processed, which makes possible the study of platelet size in a great variety of clinical conditions.[3] Within an individual, platelets are heterogeneous in size and density. Larger platelets are metabolically and enzymatically more active, and have greater prothombotic potential .[4,5]

Elevated MPV is associated with other markers of platelet activity, including increased platelet aggregation, increased thromboxane synthesis and β-thromboglobulin release, and increased expression of adhesion molecules.[6]

Higher MPV and increased PDW have been found in sepsis. It has been demonstrated thatcoagulation and platelet activation/hyperaggregation can occur in an early phase of sepsis.[7] In order to obtain a larger surface, platelets change their discoid shape to a spherical shape during activation. At the same time, pseudopodia formation occurs.

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Platelets with increased number and size of pseudopodia may affect the PDW.[8]Platelet volume is related to platelet function and activation as well.[9]

MPV is an indicator of the average size and activity of platelets. Larger platelets are younger, more reactive and aggregable. Hence, they contain denser granules, secrete more serotonin and β-thromboglobulin, and produce more thromboxane A2 than smaller platelets.[10,11,12,13]All these can produce a pro-coagulant effect and cause thrombotic vascular complications. This suggests a relationship between the platelet function especially MPV and diabetic vascular complications thus indicating changes in MPV reflect the state of thrombogenesis.[10,14]

We know that larger platelets are considered to be metabolically, enzymatically and functionally more active than the smaller platelets.[15]They contain more dense granules and hence are more potent and thrombogenic and this might be a cause for hyper-lipidemia being a pre-thrombotic state.[16]

Platelets play a pivotal role in atherothrombosis, the major cause of most unstable coronary syndromes . Central to the pathogenesis of occlusive arterial disease is the activation of platelets at sites of vascular injury via pathologically exaggerated and deregulated versions of the protective mechanisms involved in hemostasis .[17] Platelets secrete and express a large number of substances that are crucial mediators of coagulation, inflammation, thrombosis, and atherosclerosis .[18,19] Present study was designed to find out variation in platelet parameter in different clinical conditions.

Material and method:-

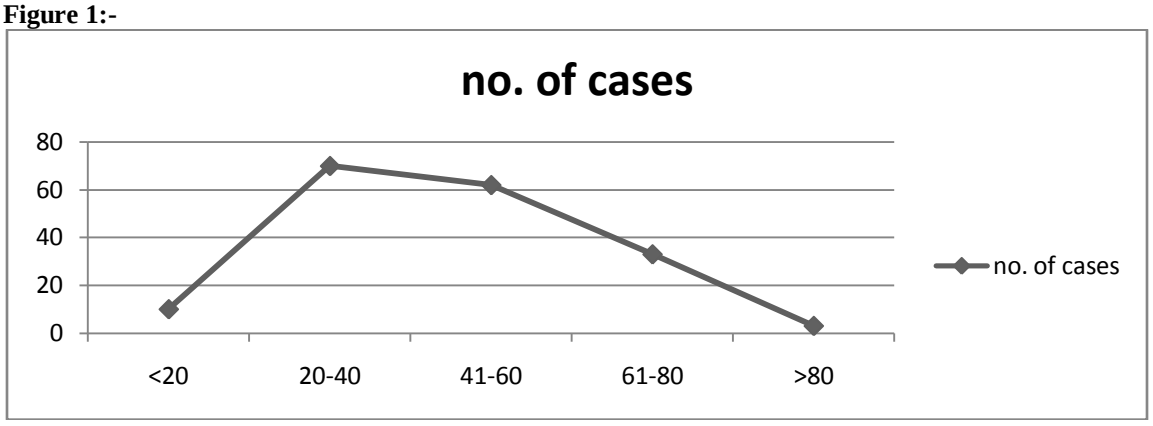
Current study was a retrospective one and it was carried out in pronounced laboratory in India .A total of 1000 subjects were searched in which 178 showed high MPV. From these 178 patients, 111 had other abnormal parameters also like high Total Count ,lipid and glucose. Rest 67 showed no abnormality hence they were used as control.

Both OPD and IPD samples were included. Sample collection for OPD patients was centralized for different sections of central laboratory. IPD samples were collected in wards, ICUs and OTs and transported to IPD sample collection centre by attendants of the respective wards.From collection centres proper samples and properly filled forms were distributed to our lab for analysis .EDTA blood samples drawn at admission of the patient were analyzed in an automated hematology analysis system that measures platelet size using aperture-impedance technology. All patient samples were processed within 2 hours after venipuncture as recommended in the literature to avoid bias due to excessive platelet swelling.The reports are validated by a pathologist. After validation these reports are printed .Reports are also being shown online so that other staffs can also see the report.Every patient is allotted a specific IP number.This number if entered in the software shows all the tests which were performed on that patients sample.The values which are high are made bold and they accompany a # and those values which were low were also bold and accompanied with *.Along with high MPV other parameters which were included are glucose,total WBC count,lipid and CKMB.(FIGURE 3)

Results:-

The present study included 1000 cases of hematology among which 178 showed high MPV(>=9).10 cases showed low MPV(<6.5).Most common age group of high MPV in our study was found to be 20-40 years.(FIGURE 1) which included 70 cases.Next commonest age group was 41-60years.Least number of cases were found in group >80 years.

Figure 1:-



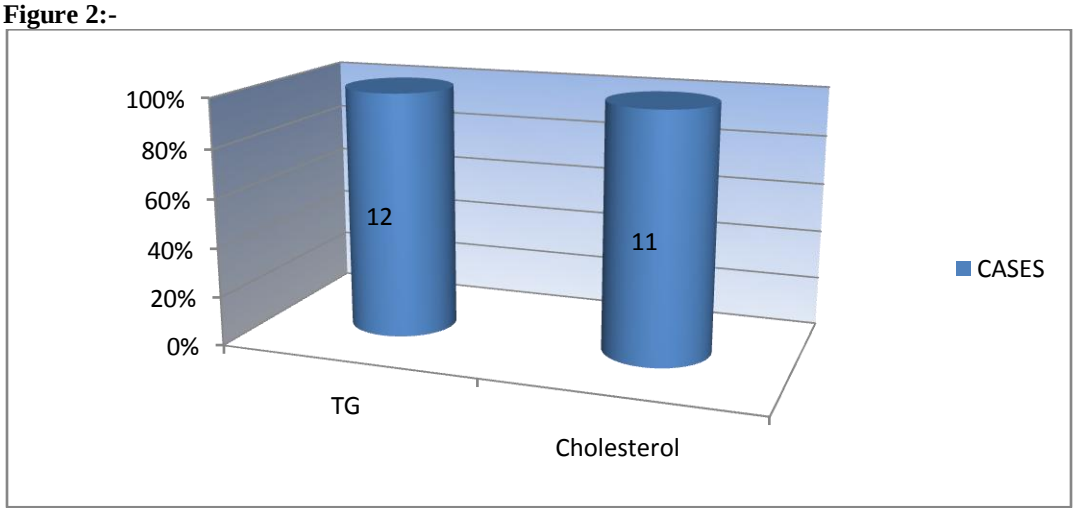
In this study it was found that males(94 cases) had slightly more increased MPV than females(84 cases).The M:F ratio came out to be 1.12.

On comparing other biochemical parameters we found that 50 (28%) cases of high MPV also showed high total WBC count.Majority of the cases showed counts above 11,500 whereas 5 cases were critically high.67 cases out of 178 showed normal total counts.

Hyperglycemia was the second parameter which was compared.It showed higher correlation than other parameters compared.63 (35.3%) cases showed hyperglycemia.6 cases were critically high.

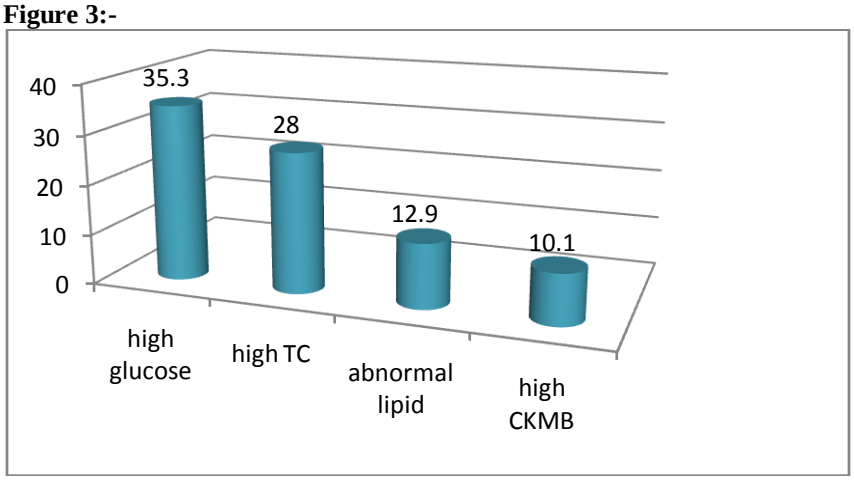
Next comes lipid profile which showed 23 cases with high levels.Different patients showed different abnormal parameters.Among these 12 patients showed high triglycerides(TG),11 showed high cholesterol.Majority of patients showed combined elevation of TG with VLDL & cholesterol with LDL.(FIGURE 2).

Figure 2:-



The last parameter compared was CKMB which was critical high in all18 patients.
67 patients however showed no biochemical abnormality.
26 patients showed both high glucose and total count.

Figure 3:-



Discussion:-

A close relationship between MPV and Cardiovascular risk factors, such as hypertension, impaired fasting glucose, diabetes, hypercholesterolemia and obesity has been demonstrated in several studies. In hypertension and diabetes, MPV is higher in patients with the progression of basic disease and the presence of target organ damage. In patients with diabetes, MPV positively correlates with microvascular complications, and severity of angiographically proven coronary artery disease. In hypertensive patients, MPV levels strongly correlate with severity of subclinical target organ damage, including carotid atherosclerosis, left ventricular hypertrophy and renal dysfunction. Hypercholesterolemia, hypertriglyceridemia and abdominal obesity are directly associated with elevated MPV. [20]

We studied 1000 cases among which 178 showed high MPV. Among these 67(37.6%) showed no abnormality hence they were used as control.

In the present study most common age group of high MPV in our study was found to be 20-40 years.However DOMAGOJ MARKOVIé et al found median age to be 65 years.[21] Georg Slavka et al found most common age range to be 34 to 65 years.[22]

According to our study males were slightly more affected than females.M:F ratio came to be 1.12. Georg Slavka et al found female preponderance with F:M ratio:1.3:1.On the other hand DOMAGOJ MARKOVIé et al found male (65.7%) preponderance in their studies.

In our study we found that 50 cases(28%) of high MPV also showed leucocytosis. Majority of the cases showed counts above 11,500 whereas 5 cases were critically high.67 cases out of 178 showed normal total counts.Guclu Eet al in his studies found high correlation between MPV and sepsis.Out of 288 cases 145(50%) showed high MPV with sepsis.[23]

Our study showed higher correlation between hyperglycemia and MPV than other parameters compared.63 (35.3%) cases showed hyperglycemia out of which 6 cases were critically high.Thomas Alex found that diabetic patients showed high MPV values.[24] This also agreed with the findings seen in studies done by Hekimsoy *et al*.[25] And Demirtunc *et al*.[26]

According to present study lipid profile was high in 23 cases i.e12.9% .Different patients showed different abnormal parameters.Among these 12 patients showed high triglycerides(TG),13 showed high cholesterol,14 showed high LDL and 10 showed high VLDL.Majority of patients showed combined elevation of TG with VLDL & cholesterol with LDL. Grotto et al also found that MPV and PDW were significantly higher in dyslipidemic patients than in controls.[27]RashiKhemka et also found similar results in their studies.[28]

Lastly we measured CKMB which was critical high in all18 patients and constituted 10.1% of cases. Murat Civan et al in his studies also found positive correlation between highMPV and CKMB.[29]

Conclusion:-

A close relationship between MPV and Cardiovascular risk factors, such as hypertension, impaired fasting glucose, diabetes, hypercholesterolemia and obesity has been demonstrated in several studies. In hypertension and diabetes, MPV is higher in patients with the progression of basic disease and the presence of target organ damage. In patients with diabetes, MPV positively correlates with microvascular complications, and severity of angiographically proven coronary artery disease. In hypertensive patients, MPV levels strongly correlate with severity of subclinical target organ damage, including carotid atherosclerosis, left ventricular hypertrophy and renal dysfunction. Hypercholesterolemia, hypertriglyceridemia and abdominal obesity are directly associated with elevated MPV.

In the present study our results showed positive correlation between high MPV,leucocytosis, hyperglycemia, hyperlipidemia and high CKMB. Hence high MPV can be used as an early indicator of these parameters.

References:-

1. Wiwanitkit V. Plateletcrit, mean platelet volume, platelet distribution width: its expected values and correlation with parallel red blood cell parameters. ClinApplThrombHemost. 2004;10(2):175-178.
2. Boos CJ, Lip GY. Assessment of mean platelet volume in coronary artery disease - what does it mean? Thromb Res. 2007;120(1):11-13.
3. Osselaer J, Jamart J, and Scheiff J. Platelet distribution width for differential diagnosis of thrombocytosis; *Clinical Chemistry* 1997;43(6): 1072-76
4. Karpatkin S. Heterogeneity of human platelets. II. Functional evidence suggestive of young and old platelets. J Clin Invest. 1969;48:1083–1087.
5. Kamath S, Blann AD, Lip GY. Platelet activation: assessment and quantification. Eur Heart J.2001;22:1561–1571.
6. Bath PM, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis. 1996;7:157–161.
7. Becchi C, Al Malyan M, Fabbri LP, Marsili M, Boddi V, Boncinelli S. Mean platelet volume trend in sepsis: is it a useful parameter? Minerva Anestesiol. 2006;72(9):749-56.
8. Vagdatli E, Gounari E, LazaridouE,Katsibourlia E, Tsikopoulou F, LabrianouI.Platelet distribution width: a simple, practical and specific marker of activation of coagulation. Hippokratia. 2010;14:28-32.
9. Bilici S, Sekmenli T, Göksu M, Melek M, Avci V. Mean platelet volume in diagnosis of acute appendicitis in children. Afr Health Sci. 2011;11:427-432.
10. Hekimsoy Z, Payzinb B, Ornek T, Kandogan G. Mean platelet volume in Type 2 diabetic patients. J Diabetes Complications 2004;18:173-6.
11. Colwell JA, Nesto RW. The platelet in diabetes-focus on prevention of ischemic events. Diabetes Care 2003;26:2181-8.
12. Ateº O, Kiki I, Bilen H, Keleþ M, Koçer I, Kulaçoðlu DN *et al* . Association of Mean Platelet Volume With The Degree of Retinopathy in Patients with Diabetes Mellitus. Eur J Gen Med 2009;6:99-102.
13. Chang HA, Hwang HS, Park HK, Chun MY, Sung JY. The Role of Mean Platelet Volume as a Predicting Factor of Asymptomatic Coronary Artery Disease. Korean J Fam Med 2010;31:600-6.
14. Bae SH, Lee J, Roh KH, Kim J. Platelet activation in patients with diabetic retinopathy. Korean J Ophthalmol 2003;17:140-4.
15. Corash L, Tan H, Gralnick HR. Heteroge-neity of human whole blood platelet sub-populations. I. Relationship between buoyant density, cell volume and ultra-structure. Blood 1977;49:71-87.
16. Jindal S, Gupta S, Gupta R, Kakkar A, Singh HV, Gupta K et al. Platelet indices in diabetes mellitus: indicators of diabetic microvascular complications. Hematology 2011;16:86-9.
17. Davi G, Patrono C. Platelet activation and atherothrombosis. N Engl J Med. 2007;357:2482–2494.
18. Coppinger JA, Cagney G, Toomey S, Kislinger T, Belton O, McRedmond JP, Cahill DJ, Emili A, Fitzgerald DJ, Maguire PB. Characterization of the proteins released from activated platelets leads to localization of novel platelet proteins in human atherosclerotic lesions. Blood. 2004;103:2096–2104.
19. Gawaz M, Langer H, May AE. Platelets in inflammation and atherogenesis. J Clin Invest.2005;115:3378–3384.
20. DomagojMarković ,VedranCarević, DamirBonacin, BrankaPaukovićSekulić, Ada Sapunar, DamirFabijanic. Correlation between mean platelet volume and total risk of cardiovascular disease. SIGNA VITAE 2013; 8(2): 49 – 55.

21. DOMAGOJ MARKOVIé • VEDRAN CAREVIé • DAMIR BONACIN • BRANKA PAUKOVIéSEKULIé • ADA SAPUNAR • DAMIR FABIJANIé. Correlation between mean platelet volume and total risk of cardiovascular disease. SIGNA VITAE 2013; 8(2): 49 – 55.
22. Georg Slavka, Thomas Perkmann, HelmuthHaslacher, Stefan Greisenegger, Claudia Marsik, Oswald F. Wagner, Georg Endler. Mean Platelet Volume May Represent a Predictive Parameter for Overall Vascular Mortality and Ischemic Heart Disease. (ArteriosclerThrombVasc Biol. 2011;31:1215-1218.)
23. Guclu E, Durmaz Y, Karabay O. Effect of severe sepsis on platelet count and their indices. African Health Sciences 2013; 13(2): 333 – 338.
24. Thomas Alex Kodiatte, Udaya Kumar Manikyam, SurakshaBellurRao, Thej MothakapalliJagadish, Madhavi Reddy, Harendra Kumar Malligere Lingaiah, and Venkataswamy Lakshmaiah. Mean Platelet Volume in Type 2 Diabetes Mellitus. J Lab Physicians. 2012 Jan-Jun; 4(1): 5–9.
25. Hekimsoy Z, Payzinb B, Ornek T, Kandogan G. Mean platelet volume in Type 2 diabetic patients. J Diabetes Complications. 2004;18:173–6.
26. Demirtunc R, Duman D, Basar M, Bilgi M, Teomete M, Garip T. The relationship between glycemic control and platelet activity in type 2 diabetes mellitus. J Diabetes Complications. 2009;23:89–94.
27. Grotto HZ, Noronha JF. Platelet large cell ratio (P-LCR) in patients with dyslipidemia. Clin Lab Haematol 2004;26:347-9.
28. Khemka R, Kulkarni K. Study of relationship between Platelet Volume Indices and Hyperlipidemia. Annals of Pathology and Laboratory Medicine. 2014;1(1):8-14.
29. Murat Civana, SedatÖzcanb, Murat Ziyrekc, FatmaCand, Murat Gülbaran.
30. Relationship between cardiovascular evaluation data on admission and secondary adverse cardiac event rate after acute coronary syndrome.
31. J. Exp. Clin. Med., 2014; 31:81-85.